A REPORT ON FIVE SURGICAL CASES

FROM THE WESTERN GENERAL HOSPITAL, EDINBURGH

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Pattison Prize in Clinical Surgery

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INTRODUCTION

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An eight week student attachment in general surgery is clearly insufficient to turn ignorance into surgical expertise, but the five cases which I would like to present each demonstrated to me a point in the "art of surgical practice". All five stimulated me to learn their condition in more detail and as some of the earliest surgical cases I have encountered, they will always be the face I associate with their particular condition.

Firstly, I would like to present two cases of colorectal carcinoma. My first case is that of a particularly aggressive tumour and the second a case of multiple tumours of the large bowel.

Secondly, I would like to present and discuss a case which demonstrated clearly to me the problems that can arise as a result of relatively simple surgery, in this case a cholecystectomy.

Lastly, I encountered two cases which, although straightforward in nature, taught me the correct method of examination of the groin and its differential diagnosis, and the current thinking on testicular tumours.

PART I

COLORECTAL CARCINOMA

Case 1

Mr. R.M. is a 51 year old man who presented to the medical unit with a four week history of:

1. epigastric and right loin pain;
2. liver tenderness;
3. vomiting and diarrhoea;
4. anorexia;
5. weight loss;
6. lethargy.

Initially, he noticed a constant, "aching" pain across his epigastrium which became gradually worse and was associated with some right loin discomfort and tiredness. In addition, his general practitioner detected tenderness over his liver, especially the right lobe. Symptoms eased over the next ten days under conserative management while his general practitioner conducted several investigations.

An out-patient oral cholecystogram failed to demonstrate any gall-stones to account for his liver tenderness but clinical chemistry showed his liver enzymes to be raised:

			20.8.87	27.8.87	(Normal Values)
plasma lactate dehydrogenase	[LDH]	u/l	1020	1143	(72-395)
plasma alkaline phosphatase	[alk. phos.]	u/l	152	168	(30-140)
plasma aspartate aminotransferase	[AST]	u/l	32	34	(9-52)
plasma bilirubin	[bili]	μmol/l	12	10	(3-14)

Mr. R.M.'s antihypertensive medication was subsequently changed from Enalapril (10mg daily) to Atenolol (100mg daily), in case this was the cause of his deranged liver function tests, and he was referred for a medical consultation.

At medical out-patients, three weeks after becoming ill, he complained of worsening epigastric pain which radiated down his right loin. He had become unable to eat a large meal without feeling nauseous, and often vomiting. The vomitus contained only fluid and bits of food, with no frank blood or "coffee-grounds". The edge of his liver could be palpated at two finger-breadths below the costal margin and it was now diffusely tender. No other masses were detected in his abdomen.

His liver enzymes were found to be still elevated:

		31.8.87
LDH	u/l	1232
alk. phos.	u/l	36
AST	u/l	9
bili	μmol/l	166

Virology tests conducted at that time later showed Mr. R.M.'s liver complaint to be unrelated to viral infection by hepatitis A, hepatitis B, cytomegalovirus or Epstein-Barr virus.

An upper abdominal ultra-sound scan was performed as an out-patient and revealed an irregular diffuse

abnormality of the echo pattern in the left lobe of the liver which was rather non-specific in appearance. The right lobe, gall-bladder and pancreas all appeared normal.

Mr. R.M. was admitted to the medical unit on the 5th of September for further investigation.

On admission, Mr. R.M. reported that his epigastric pain was becoming progressively worse. He now felt extremely nauseous after a meal, vomiting whatever he had eaten about an hour later, although still without any signs of blood in the vomitus. His appetite had become very poor and he had lost a stone and a half in weight over the past four weeks. Recently, he had developed a loosening of his stools, associated with an increase in frequency of bowel-movements but wihout frank blood or malena. He was feeling extremely tired.

In 1984, Mr. R.M. developed a sudden onset of renal colic. Subsequent investigation revealed high blood pressure and he was discovered to have a small left kidney. This left kidney, which was contributing only 15% of renal function, was supplied by two arteries, one of which was considered to be slightly stenosed. The subsequent management of his hypertension by oral medication had been uncomplicated to date although it was

changed from Enalapril to Atenolol shortly before admission as precaution against an unusual drug reaction to Enalapril. This was the only medication he was receiving.

His only other admission to hospital was for appendicectomy as a child. He did not know of anything to which he was allergic.

Mr. R.M., an electronic engineer and married with three children, drank alcohol infrequently and although a smoker since his late teens had stopped six years previously when he had been smoking approximately 50 cigarettes daily. There was no family history of liver disease or other serious illness, and systematic enquiry did not reveal any new information about his condition.

On examination he was again found to have a tender enlarged liver which was palpable two finger-breadths below the costal margin. He had no systemic signs of liver disease. No other abnormality was detected during the examination and he was normotensive. In particular, his stools were negative when tested for faecal occult blood.

Clinical chemistry again revealed abnormal liver function tests:

		7.9.87
LDH	u/l	1198
alk. phos.	u/l	210
AST	u/l	54
bili	μmol/l	13

However his other blood chemistry was normal, including the amylase level. His erythrocyte sedimentation rate was raised at 25 mm in 1 hour (normal value less than 5) but although his prothrombin time was slightly elevated at 1.3:1, his other haematology tests were not deranged.

Further virology tests did not reveal any viral infection to account for Mr R.M.'s condition and bacteriological examination of his stools, performed in view of his slight disturbance of bowel movements, did not detect any abnormality. Culture of a mid-stream specimen of urine was also negative and his electrocardiograph and chest X-ray were normal.

Tests for connective tissue disease revealed a slight increase in C-reactive protein but anti-nuclear factor titres were normal.

On the 10th of September, Mr. R.M. had a double contrast barium meal which did not demonstrate any abnormality and the following day a repeat ultrasound scan and biopsy of his liver was performed. This again showed abnormality of the liver, particularly in the mid-line and left lobe. Under local anaesthetic a tru-cut biopsy was taken from this area, which produced a small amount of friable tissue. Consequently, a number of fine needle aspirates were taken and sent for cytological examination.

The aspirate revealed no evidence of active

inflammation with minimal fatty change in the hepatocytes. There were no signs of cholestasis. The biopsy, however, was too small for definitive diagnosis.

On the 16th of September clinical chemistry reported that the isoenzymes of the raised alkaline phosphatase were partly normal liver enzymes and partly abnormal enzymes, also of hepatic origin. On the same day, rigid sigmoidoscopy was carried out. This showed normal mucosa in the rectum and lower sigmoid colon with some loose stools. However, at 12 centimetres, a 5 milimetre area of abnormal mucosa was found which appeared to be a sessile polyp. Pathology detected features of mild, non-specific chronic inflammation surrounding a metaplastic polyp. Tests for faecal occult blood were again negative.

The following day, liver scintigraphy revealed a large liver with multiple areas of focal replacement, highly suggestive of metastatic involvement [see Figure 1].

Mr. R.M. was sent home until the following week when renal scintigraphy was carried out. This demonstrated the small left kidney, as before, which was consistent with a left renal artery stenosis [see Figure 2].

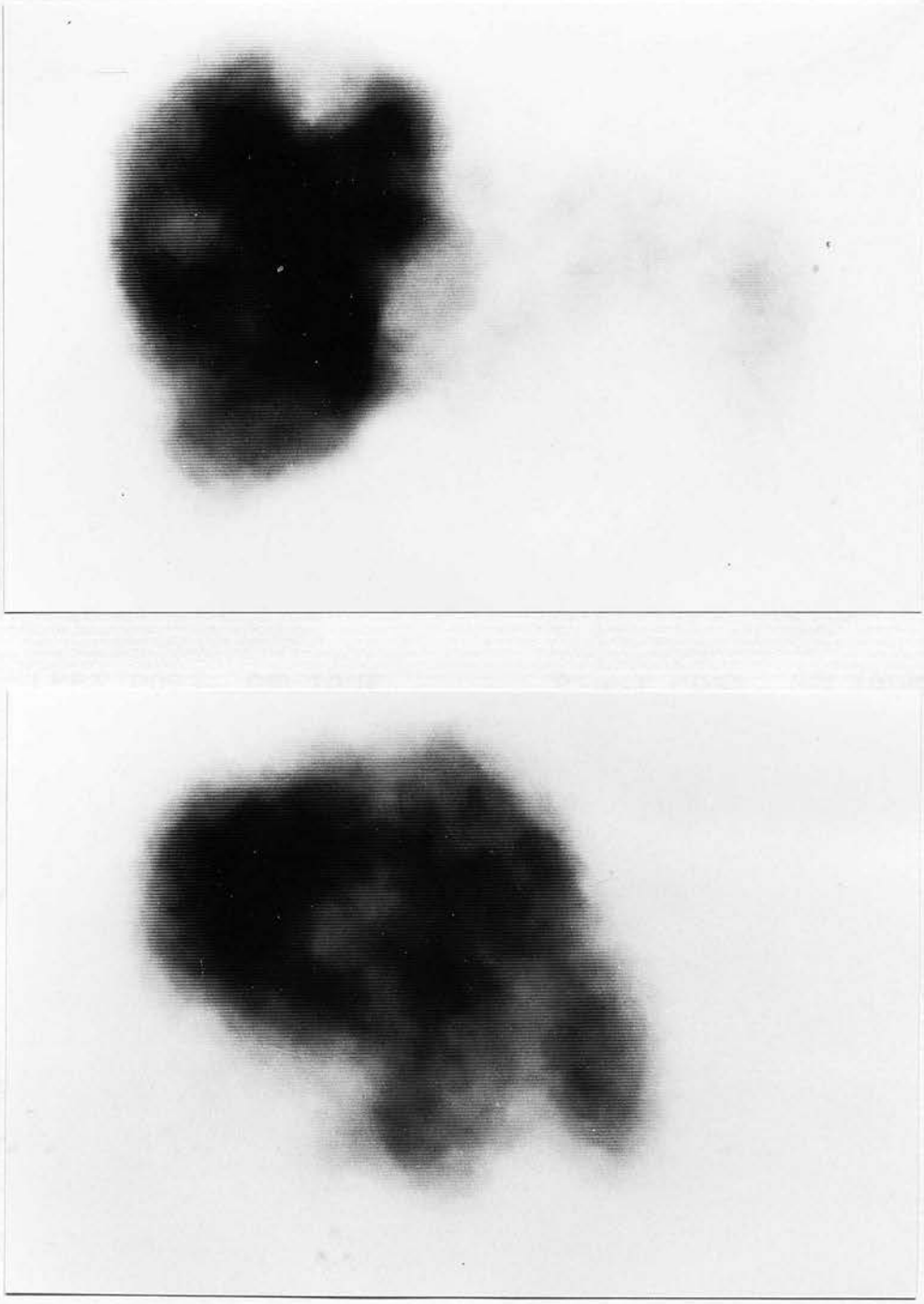

Figure 1: Liver scan showing large liver with multiple areas of focal replacement

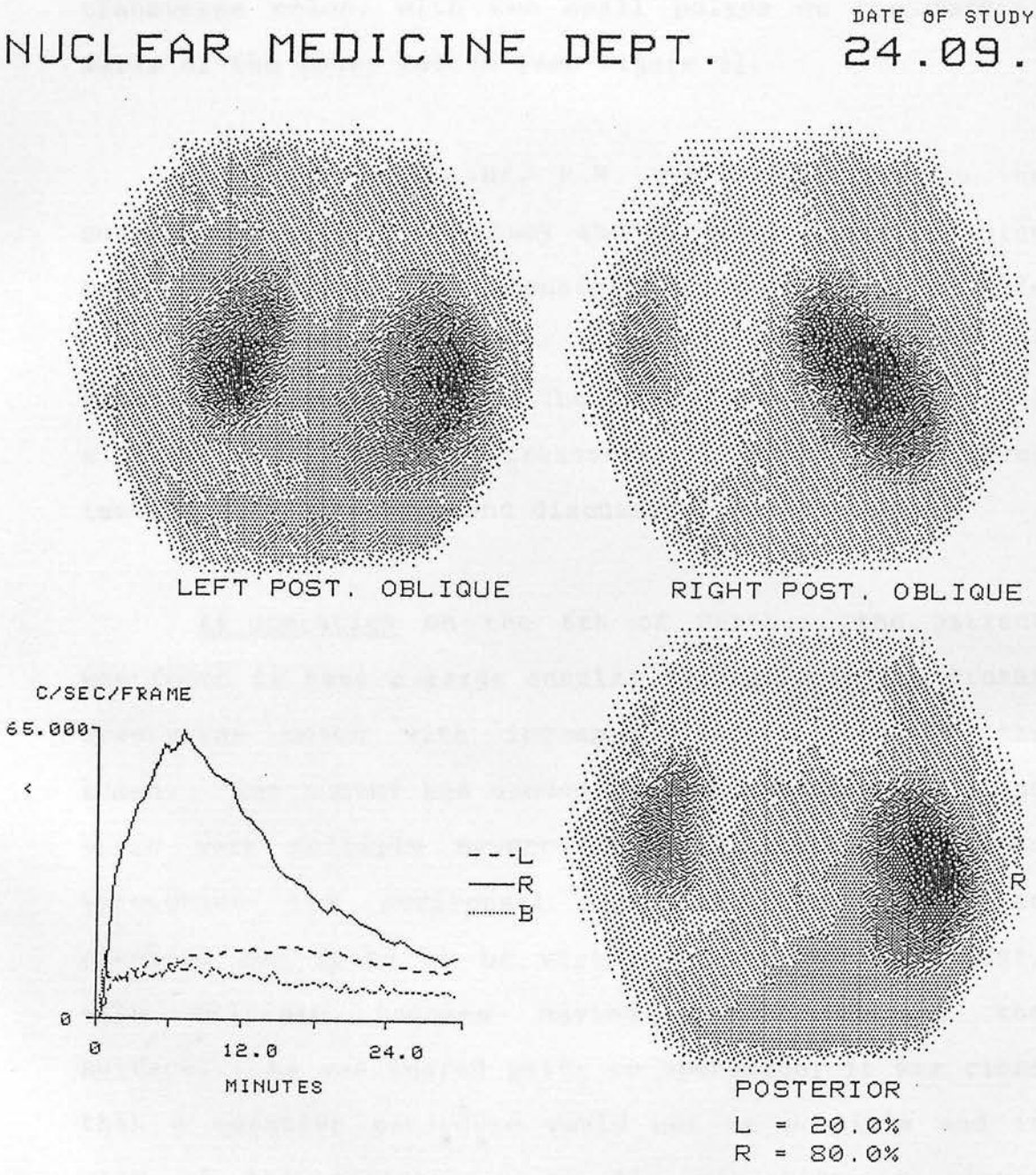

Figure 2: Renal scan showing a small left kidney which is contributing less than 20 per cent of renal function

On the 29th of September, a barium enema revealed a large, 6 x 4 centimetre carcinoma in the distal transverse colon, with two small polyps on the lateral walls of the lower rectum [see Figure 3].

At this point, Mr. R.M. was transferred to the surgical unit for laparotomy and to avoid impending large bowel obstruction. On transfer, his progressive rise in liver enzymes [see Table 1] and past history of hypertension were noted. The barium enema films showing a large tumour in his transverse colon and the liver isotope scan were seen and discussed.

At operation on the 6th of October, the patient was found to have a large annular carcinoma in the distal transverse colon with incomplete obstruction of the lumen. The tumour had eroded through the bowel wall and there were multiple mesenteric secondaries and spread throughout the peritoneal cavity. The liver was examined and found to be virtually replaced by tumour, with multiple nodules having eroded through the surface. As was feared prior to operation, it was clear that a curative procedure would not be possible and in view of the seriousness of the situation, a simple transverse colon to sigmoid colon, side to side anastomosis was carried out to by-pass the tumour and achieve the best palliation under the circumstances.

A formal wedge liver biopsy was taken and

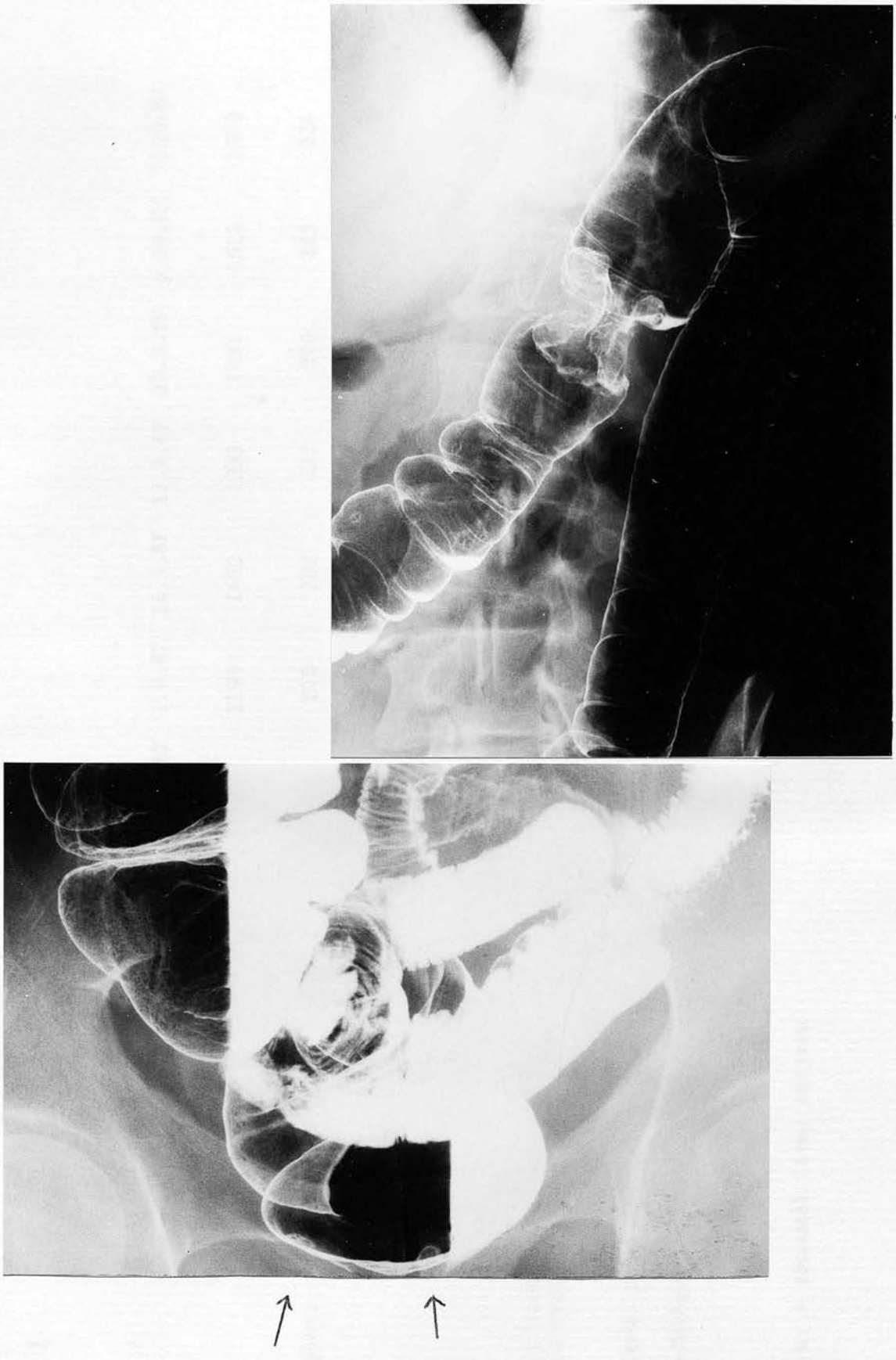

Figure 3: Barium Enema showing a carcinoma in the distal transverse colon and two polyps in the rectum

Table 1

		20.8.87	27.8.87	31.8.87	7.9.87	14.9.87	17.9.87	28.9.87	5.10.87	7.10.87
LD	u/l (72-395)	1020	1143	1232	1198	1452	1398	1491	1512	1570
alk. phos.	u/l (30-140)	152	168	166	210	202	218	290	285	224
AST	u/l (9-52)	32	34	36	54	50	54	88	60	90
bili	µmol/l (9-52)	12	10	9	13	12	12	15	17	15
gamma-glutamyl transferase	u/l (8-49)					124		219		
alk. phos. isoenzymes					*					
alpha-fetoprotein	u/ml (2-6)					3		3		

* liver + abnormal liver enzymes

pathology later confirmed mucin-secreting adenocarcinoma, entirely consistent with colonic origin.

Mr. R.M. made a good recovery from the procedure and was discharged to North Berwick Hospital on 16th of October, prior to going home. With all the facts available, the situation was discussed with the patient in the presence of his wife, and the seriousness of the prognosis explained.

Subsequent management was co-ordinated by his general practitioner, with advice from the medical director at St. Columba's Hospice. Mr. R.M.'s main problem became the pain he was experiencing at night and the resulting disturbance in sleep. Consequently, he required an increasing quantity and strength of analgesia. In addition, he was anorexic and found difficulty coming to terms with his condition and transparently short prognosis.

Sensitive counselling and careful management of his medication by those caring for him, allowed him to die peacefully in his local hospital on the 27th of December. Less than twenty weeks had passed since the initial onset of his symptoms and presentation to his general practitioner.

Discussion

Adenocarcinomas of the colon and recum are common, potentially curable and possibly preventable. Other malignancies in this region, such as lymphoma, sarcoma and carcinoid, are very rare.

Cancer of the large bowel is the second commonest malignancy in the United Kingdom after lung cancer. In this country, 16,000 deaths are reported annually, 10,000 of these are in the colon and the other 6,000 in the rectum.[1] Approximately 12.5 per cent of all cancer deaths are due to colorectal carcinoma. Men appear to be slighly more at risk from rectal cancer while women are more likely to develop colon cancer. The indicence for both increases with age, the average age at diagnosis being between 60 and 65 years.

Epidemiology suggests that both environmental and genetic factors are important in colorectal cancer.[2] Incidence is higher in northern Europe and North America but also those in low-risk areas, such as Japan, who adopt a "westernised diet".

Heavy meat-eating groups in low risk areas have a high incidence, and conversely, vegetarians in high risk areas show a decreased incidence. It has been postulated that carcinogens may be produced by bacterial metabolism of the bile salts, or other sterols derived

from animal fat and that the slow colonic transit associated with a low fibre diet allows greater contact of these carcinogens with the bowel mucosa. Ethanol has also been associated with colon carcinogenesis.

Genetic factors are implicated by the increased incidence of cancer within families: 15 per cent of siblings and 10 per cent of children of patients with colorectal cancer develop the disease. Familial polyposis is an extreme example of the importance of genetic factors. Also, other factors must be involved to explain the increased incidence in association with breast, ovarian or prostatic cancer.

In effect, the suggested hypothesis for the aetiology of colorectal cancer is that predisposition to adenomas occurs if a recessive gene is inherited from each parent, and that dietary factors result in a changed bacterial flora, with an increase in carcinogen production, which promotes adenoma formation and eventually malignant transformation.[3]

From a clinical viewpoint, the sequence of events is usually slow, allowing removal of the precancerous lesions. Also most carcinomas of the large intestine are, at least, moderately well differentiated and present before they have metastasised.[4,5]

The majority of bowel carcinomas occur distally in

the rectosigmoid. The typical carcinoma is a polypoid mass with central ulceration and irregular easily bleeding edges, which may spread to become a stricture. Initially, colorectal carcinoma spreads by local invasion so that its prognosis is good when confined to the bowel wall. Involvement of the larger veins results in early spread to the liver and poor prognosis, although metastases are occasionally single and resectable. Spread by lymphatics occurs first to nodes next to the tumour and only later to nodes lying more centrally in the mesentery. This gradual progression makes complete resection of large bowel carcinoma more feasible.[6]

In 1932, Dukes formulated a staging classification for resected carcinoma of the rectum, which in turn was applied to colonic cancers. In this, the extent of local spread and presence or absence of lymph node metastases was related to five-year-survival [see Figure 4].

Classification may also be made according to histological grade - well differentiated, moderately-well differentiated and poorly differentiated - but this is less accurate in predicting the prognosis.

The most common sites of metastases are liver (over 60 per cent), lung (over 50 per cent), peritoneum (15 per cent) or skeleton (15 per cent).[7]

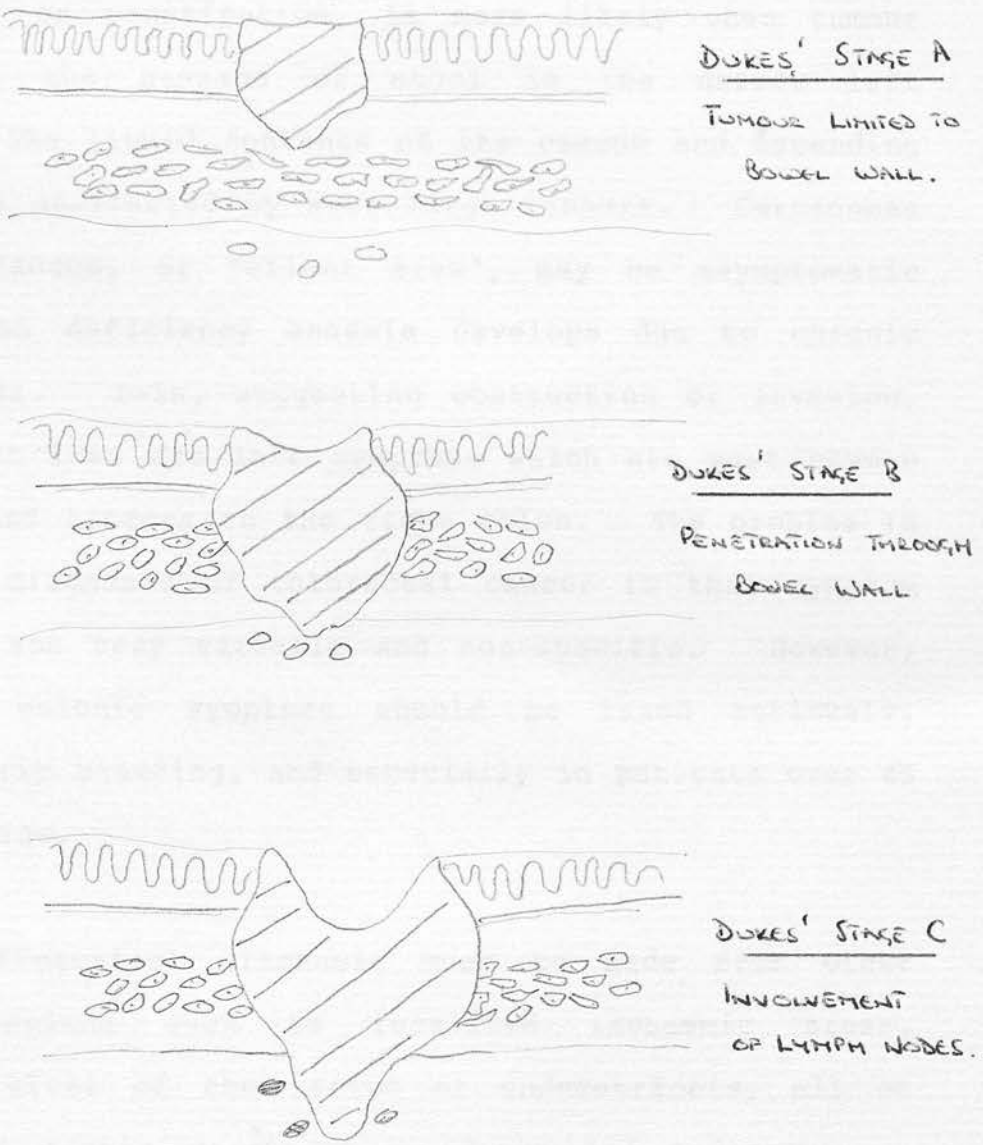

Figure 4: Duke's classification of colorectal carcinoma

The symptoms and signs of a colorectal carcinoma depend on its site. Rectal and sigmoid colon cancers usually bleed due to buffeting by solid stool. Alteration in previously regular bowel habit, both frequency or constipation, is more likely when tumour obstructs the passage of stool in the narrow left colon. The liquid contents of the caecum and ascending colon are unaffected by even large tumours. Carcinomas of the caecum, or "silent area", may be asymptomatic until iron deficiency anaemia develops due to chronic blood loss. Pain, suggesting obstruction or invasion, and weight loss are late symptoms which are most common in advanced cancers in the right colon. The problem in clinical diagnosis of colorectal cancer is that symptom patterns are very variable and non-specific. However, any new colonic symptoms should be taken seriously, particularly bleeding, and especially in patients over 45 years of age.

Differential diagnosis must be made from other benign lesions such as localised ischaemic areas, solitary ulcer of the rectum or endometriosis, all of which can mimmic carcinoma on X-ray and endoscopy. Palpable masses with radiological features simulating carcinoma are found in the caecal area due to an appendix abscess, tuberculosis or Crohn's disease, and in the sigmoid colon by diverticular disease with pericolic abscess. Strictures causing suspicious X-ray

appearances are seen in Crohn's disease, ulcerative colitis, or after ischaemic colitis. Therefore, a forceps biopsy should be taken before operating on an apparent carcinoma whenever practiable.[3]

The lack of sensitivity and specificity of serological tests such as the carcinoembryonic antigen (CEA) has been disappointing to date. It is unreliable in screening for primary diagnosis of colonic cancer but may have a place in monitoring for recurrance.[8]

The best treatment for colorectal carcinoma is excision, usually by surgical resection. Despite enthusiastic reports on radiotherapy for rectal carcinoma, this method is usually reserved for unfit patients with inoperable tumours, especially for pain relief. Chemotherapy[9] has not been shown to improve survival either in advanced disease or in patients undergoing curative surgery.

The long term results of surgery for colorectal cancer depend on the centre reporting as well as the stage and grade of tumour.[1] Specialist centres report over 90 per cent of cases as being suitable for radical or curative surgery with currected five-year survival of 50 to 60 per cent overall and nearly 100 per cent for Dukes' A cases. District hospitals quote only 50 per cent as being suitable for radical surgery, and even so, have only a 30 per cent corrected five-year survival [see Table 2].

Table 2: Prognosis and Duke's classification for colorectal cancer

Duke's Classification	Cases resected %	Corrected Five-year survival %
A	15	95-100
B	40	65-75
C_1	35	30-40
C_2	10	10-20

Further improvement in survival figures must rest on public attitude towards early reporting of bleeding or bowel symptoms and their prompt referral for investigations.

Summary and Conclusions

It is clear from his history that Mr. R.M. had a particularly aggressive tumour of the colon. He had an extremely short history of only four weeks when he initially came into hospital and it consisted of vague, non-specific symptoms. Even his signs on examination were not specific; notably, he was found to be negative for faecal occult blood on admission. It is difficult to say how this man's management could have been changed to improve significantly his survival.

However, this case also highlights the importance of proper care for the terminally ill so that they may

die peacefully and with some dignity. In particular, proper control of pain relief may allow a patient to die comfortably.

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Case 2

Mr. C.C. is a 76 year old man who presented to the surgical out-patients clinic at a routine follow-up appointment.

In 1975, he presented with a six month history of epigastric pain, occurring immediately after meals and accompanied in the last month by nausea, vomiting and flatulence. Barium meal and endoscopy revealed a para-oesophageal hernia complicated by gastric volvulus.

At operation, a Nisson's fundoplication was carried out with the stomach being fixed to the epigastric peritoneum by the fixation operation of Boerema, with crural repair below the diaphragm.

In 1984, he was admitted to hospital for T.U.R.P. to relieve increasingly troublesome prostatic symptoms. The histology of resected portions showed a very low grade tumour to be present.

In 1985, Mr. C.C. developed severe constipation and bleeding from the rectum. He was admitted to hospital and found to be strongly positive for faecal occult blood. There was no mass palpable in his abdomen and his abdominal examination was otherwise unremarkable. Flexible colonoscopy revealed only a few small polyps in the distal ascending and transverse colon

but barium enema demonstrated a large filling defect in the ascending colon which was most likely a carcinoma [see Figure 5]. Liver ultra-sound scan and chest X-ray were normal and his liver enzymes were not raised.

Subsequently, a right hemicolectomy was performed with a side-to-side ileo-transverse anastomosis. Pathology of the tumour specimen revealed a moderately differentiated adenocarcinoma of Duke's stage B [see Figure 6]. Seven small polyps were also discovered in the resected colon, near to the tumour mass.

He was reviewed during 1986 and during tha time received a prostatectomy for worsening prostatic symptoms, in view of the previous histology from his T.U.R.P.

In 1987, Mr. C.C. was referred by his General Practioner to the surgical out-patients clinic with a right para-median incisional hernia. On confirmation, his admission was arranged along with a routine follow-up barium enema and liver scan. There were no symptoms or signs to suggest any recurrance of his colonic carcinoma.

However, despite being asymptomatic, a carcinoma of the descending colon was discovered at Barium enema [see Figure 7] and he was admitted urgently to the surgical unit. Fortunately, the liver ultra-sound scan and liver function tests demonstrated no abnormality.

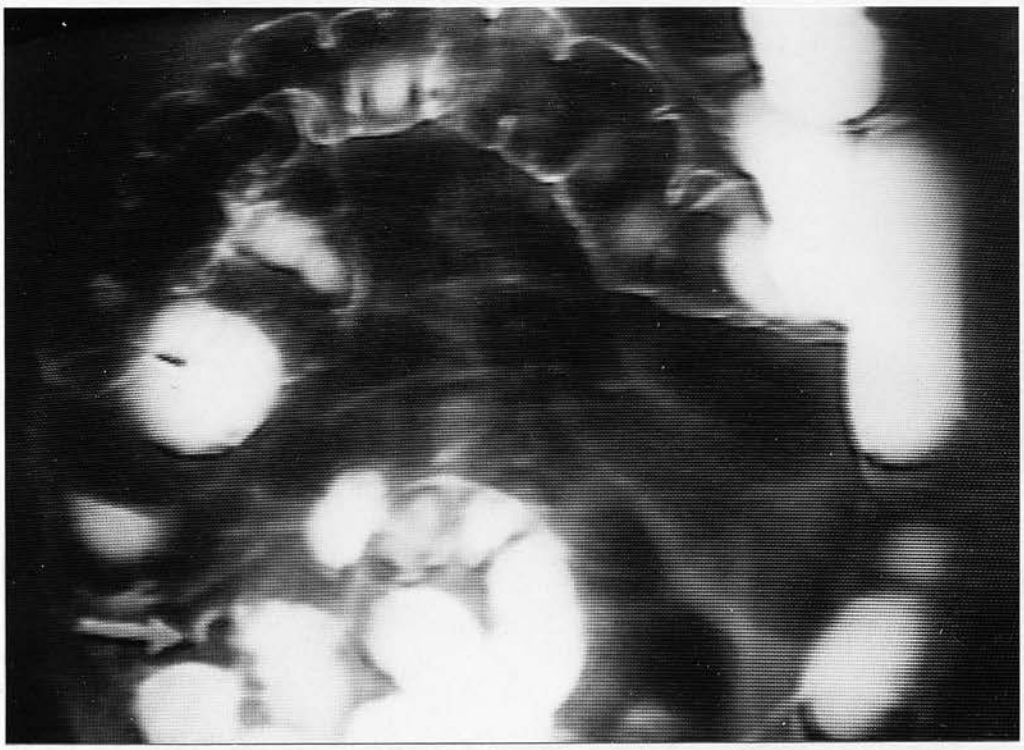

Figure 5: Barium Enema showing large filling defect in the ascending colon

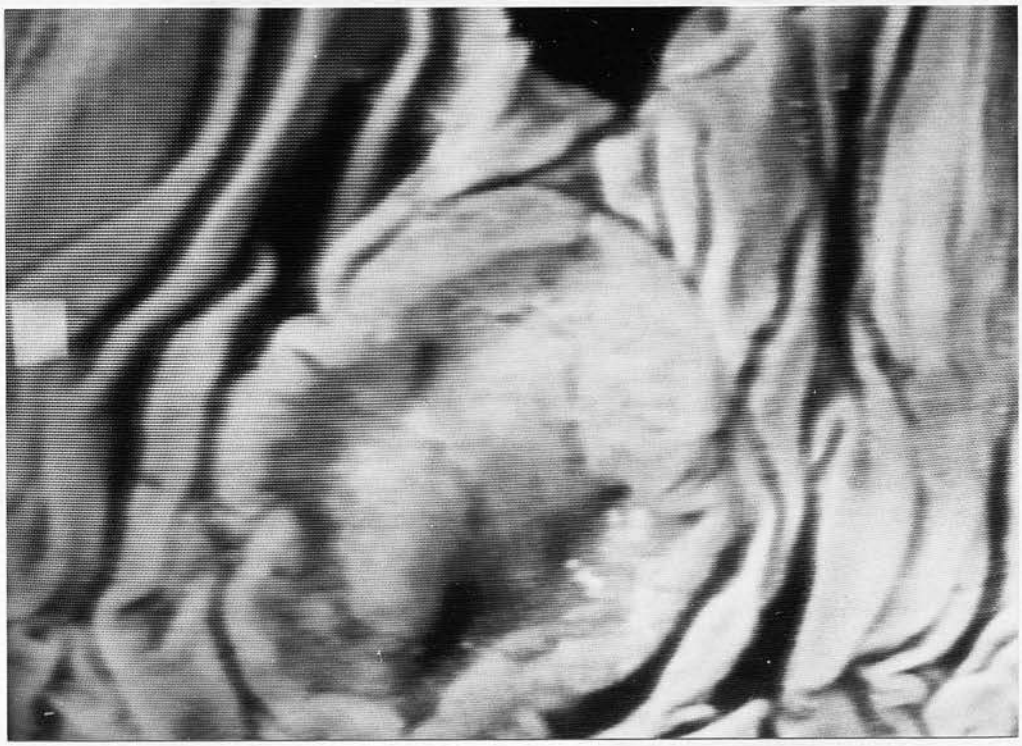

Figure 6: Tumour mass removed from ascending colon

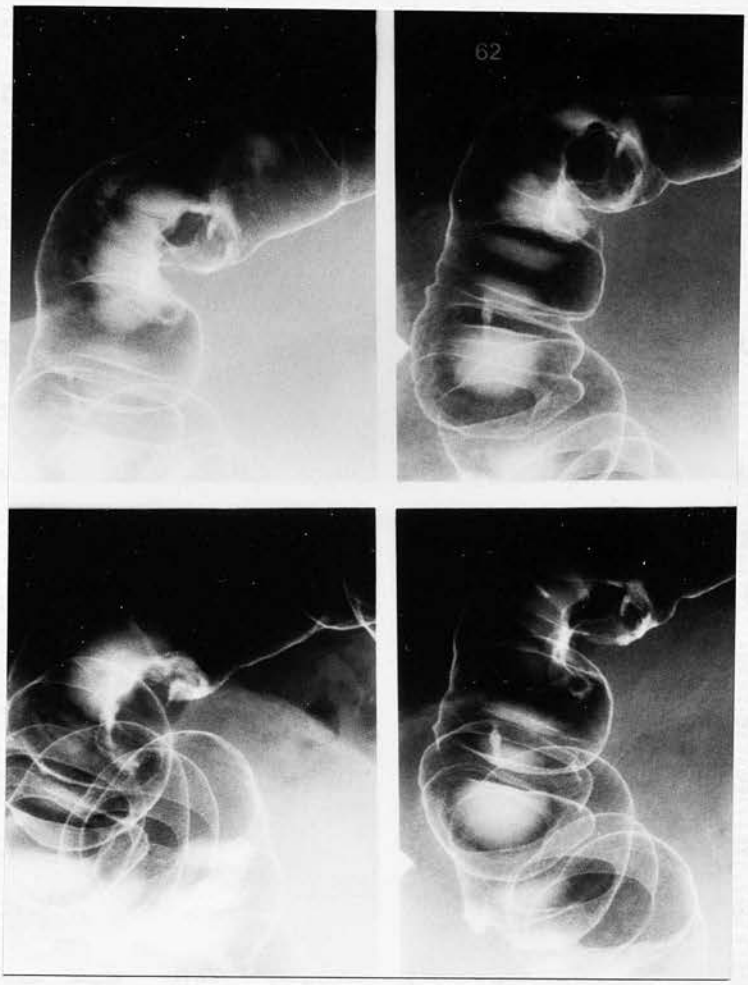

Figure 7: Barium enema showing carcinoma in descending colon

Mr. C.C. was admitted on the 29th September and flexible sigmoidoscopy subsequently revealed an adenocarcinoma at 40 centimetres, and two accompanying rectal polyps.

At operation on the 1st of October, the peritoneal cavity was opened and a flexible sigmoidoscope used to visualise the mass which could be felt through the wall of the lower descending colon. This also revealed a number of polyps and, in view of the likelihood of a "field of malignancy", it was decided that all of the remaining colon should be removed.

With some difficulty, due to adhesions resulting from the previous surgery, the transverse colon was found and its junction to the terminal ileum identified and freed. The left colic artery was tied and the splenic flexure of the colon freed from a deep position by working towards it laterally along the transferse colon and superiorly up the descending colon. The remaining vessels supplying the colon were tied and divided and the colon, stretching from ileo-colic anastomosis to rectum, formally resected. The anti-mesenteric border of the terminal ileum was then joined to the rectal stump by side-to-side anastomosis.

In Pathology, the normal mucosa of the resected colon was seen to be interrupted by a flat ulcerated mass [see Figure 8] and two sessile polyps [see Figure 9]. A moderately well-differentiated adenocarcinoma of the

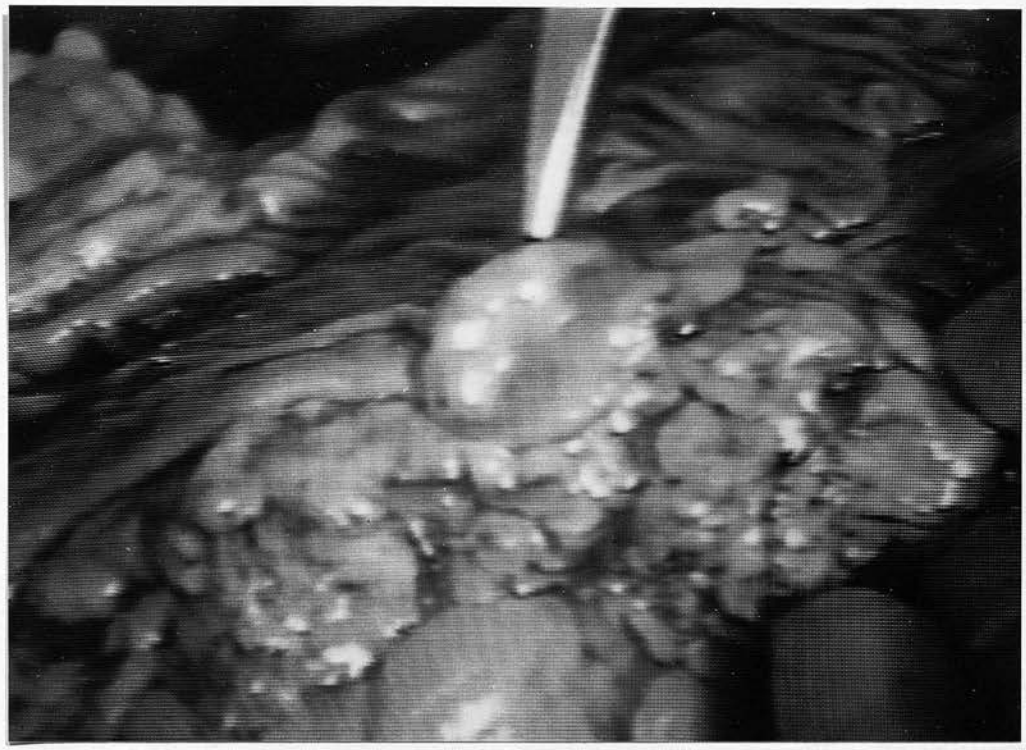

Figure 8: Ulcerated mass in resected colon

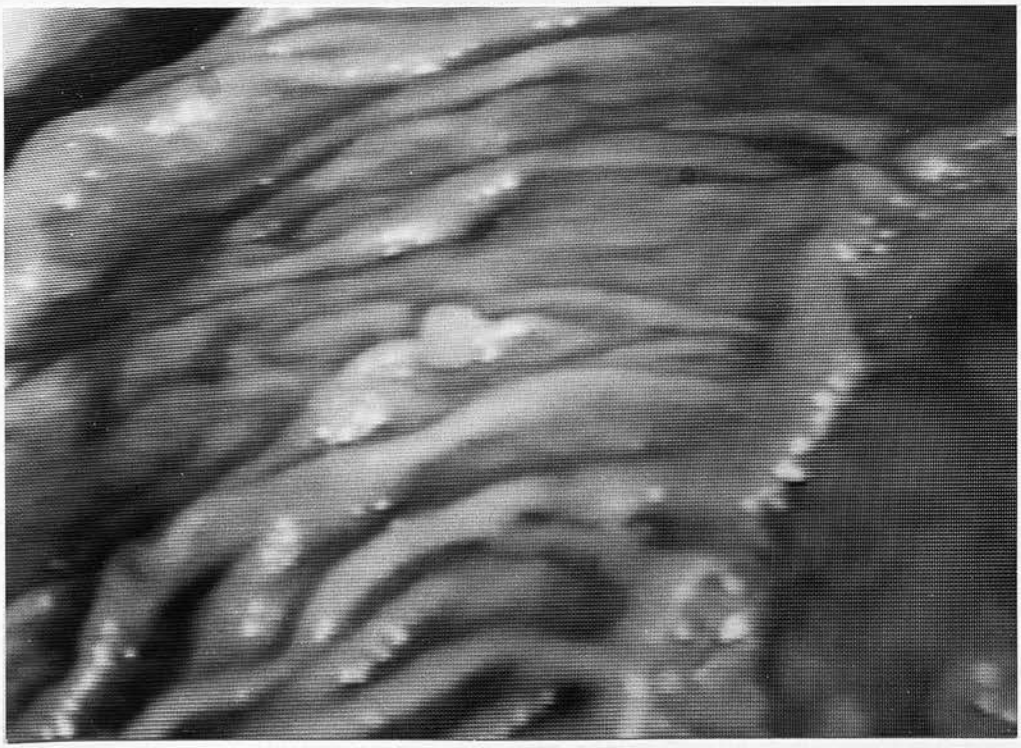

Figure 9: Sessile polyp near to tumour

large bowel was seen on histology. The tumour was seen to be inflitrating the submucosa extensively and focally, also into muscle, but no serosal invasion was identified. There was some intra-lymphatic invasion at the base of the tumour but no inflitration of blood vessels. Lymph nodes from paracolic tissue showed no evidence of metastatic carcinoma. The tumour was defined as a Duke's state B adenocarcinoma of the colon: grade 2 pT2 NO MO.

The two sessile polyps were seen to be simple hyperplastic polyps with some metaplastic features. There were no atypical features and no evidence of malignancy.

Mr. C.C. made a good recovery from his operation and was subsequently discharged to be followed-up in the out-patients clinic. After his operation in October of last year, Mr. C.C. has since returned to have a further three small polyps treated, which were detected at follow-up sigmoidoscopy.

Discussion

A metachronous cancer is one which occurs at a later date in a patient who has already been treated for that type of carcinoma, but at a new and distinct site: in this case, in the colorectal mucosa. Adenomatous polyps appear to be premalignant lesions which form a link in the natural history of colorectal carcinoma[1], where normal mucosa progresses to adenomatous polyp and then to carcinoma. A section of colon which contains adenomatous polyps demonstrates an inherent tendancy to malignant transformation and so to the development of multiple colorectal tumours. These may be synchonous (simultaneous) or metachronous (interval) cancers.

The incidence of multiple carcinomas in the colon is reported to range from 0.6 to 0.1 per cent.[2,3] In most reports, metachronous tumous appear to be only half as common as synchronous ones[4] although the true incidence of metachronous tumours may be underestimated: some patients fail to return for follow-up and others may die from the original cancer before the second develops or their second cancer may be undiscovered when they die.[5] A study in St. Mark's Hospital reported 3.2 per cent had multiple synchronous tumours, compared with 1.7 per cent with metachronous ones. This predominance of synchronous tumours correlates with most other reports,[2,5,6,7] although a study from Massachusetts[8]

reported that of 101 patients with multiple colorectal carcinomas treated over a 15 year period, 2.8 per cent had metachronous and 1.7 per cent synchronous cancers.

Synchronous cancers frequently occur adjacent to each other although Lasser[9] reported 16 per cent being separated by more than 20 centimetres. Some authors have described an increased incidence of synchronous tumours in the right colon[10] associated with a decreased incidence in the rectum.[4,7] It has been postulated that this might be due to multiple lesions in the rectum coalescing at an early stage to look more like a single lesion.[5]

There have been conflicting reports suggesting and disputing a decreased incidence of the first metachronous cancer in the rectum.[2,5,11,12]

Synchronous tumours are often unsuspected, especially when widely separated. The sensitivity of contrast studies may be decreased by stricture formation, or malignant obstruction, hindering the visualisation of the more proximal bowel.[13] Often polyps less than 1 centimetre in diameter may not be seen on barium enema but are found at operation.[14] Also, the surgeon may be unable to feel a polyp through the bowel wall. Thus, at operation, it is important to open any resected specimen and if there is any suspicion of polyps in the remainder of the colon, colonoscopy should be performed. However,

even colonoscopy may fail to reveal polyps located in flexures, hidden behind haustral folds or in telescoped bowel.[15]

The interval between primary tumour and metachronous tumour varies in different reports. Several large studies found average intervals varying between 8.5 and 11 years.[6,16,17]

It appears that most carcinomas developing from adenomas do so over a 10 to 15 year period, although Day and Morson suggest this transition is probably uncommon.[18] However, polyps are approximately twice as common in patients with multiple tumours compared to single tumours.[13,19] Also, polyps were reported in 42 to 75 per cent of synchronous tumours[6,7,8,11] and 51 to 60 per cent of metachronous tumours.[11,16,20]

Eight to 20 per cent of patients with multiple colorectal cancer have other malignancies[5,21] and so should be carefully investigated. According to Weir,[22] patients surviving a primary colorectal cancer have a greater chance of dying from an extra colonic neoplasm than from a second colorectal cancer.

Welch reported that the uncorrected survival rate in patients with multiple tumours is only half that of patients with single tumours[23] but Ekelund and Pihl found equal survival rates after resection of single or

synchronous tumours.[6]

Various operative approaches have been suggested to try to improve prognosis. Gruber et al[24] suggested that sub-total colectomy for tumours of the colon, and abdomino-perineal resection with left hemicolectomy for carcinoma of the rectum, would prevent most metachronous tumours. Enker and Dragacevic,[25] on the other hand, favoured the more liberal use of sub-total colectomy in patients with synchronous or second metachronous malignancies.

Peabody and Smithwick,[26] however, suggested a more selective approach: if an additional tumour was found in the same segment as the suspected single carcinoma, they performed a right hemicolectomy for proximal lesions and left hemicolectomy for descending colon tumours. Subtotal colectomy was recommended for transverse colon lesions and an extended Miles' operation for carcinoma of the rectum.

The conclusions of Wright,[27] are of relevance to patients being considered for ileosigmoidostomy. He concluded that they may totally adapt to the loss of colon, with the rectum acting as a valve at the ileum to slow transit and promote more efficient water and sodium transport from the ileum.

Summary and Conclusions

In a patient such as Mr. C.C., efforts should be aimed at surveillance of polyps in view of the common epidemiology which colon cancer may share with colon polyps. The colorectal mucosa of Mr. C.C. has shown a marked tendancy to epithelial hyperplasia and development of adenomatous polyps which may progress by malignant change to adeno-carcinomas. This "unstable" colorectal mucosa is liable to both synchronous and metachronous carcinomas. Mr. C.C.'s case demonstates the importance of follow-up screening in all patients who are known to have had colorectal carcinomas and the importance of thorough examination of the entire colon at the time of diagnosis of the first tumour.

Double contrast barium enema and colonoscopy are the most satisfactory methods for detecting colon polyps. The latter often detecting polyps not seen on barium enema films.

Welch[8] makes several points which shoud be considered when following patients such as Mr. C.C.:

(1) patients with synchronous tumours, or with a single carcinoma accompanied by polyps, have an increased risk of developing metachronous tumours;

(2) the second metachronous tumour is frequently right-sided and symptomatic by the time it is discovered;

(3) particular segments of the colon, such as the caecum, are sometimes inadequately visualised on barium enema or colonoscopy.

If the morbidity and mortality associated with multiple colorectal carcinoma is to be significantly reduced, Welch suggests that:

(1) careful palpation of the colon should take place at the time of resection of a colon cancer;

(2) patients should undergo regular physical examinations with testing for faecal occult blood;

(3) patients should be encouraged to return immediately on appearance of any new symptoms referrable to the gastrointestinal tract;

(4) regular sigmoidoscopy, and interval colonoscopy or barium enema examinations, should be directed towards detection of any new polyps or neoplasms in the remaining segments of large bowel.

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PART II

COMMON BILE DUCT INJURY

AT CHOLECYSTECTOMY

Case 3

Mrs. S.G., a 42 year old housewife, was referred to the surgical unit with a three month history of:

1. right upper quadrant and epigastric pain;
2. nausea and vomiting;
3. fatty food intolerance.

She initially presented to the medical out-patients clinic at the end of March. At that time, she was complaining of a severe gripping pain in the right upper quadrant and epigastrium. The pain she suffered was continuous but tended to vary in intensity throughout the day; it was accompanied by nausea, vomiting and fatty food intolerance, and was partially relieved by antacids. She was tender in the right upper quadrant but Murphy's sign negative, and her abdomen revealed nothing else which might indicate the cause of her pain. A differential diagnosis of peptic ulcer or cholecystitis was made.

On the 6th of April she was sent for an outpatient oral cholecystogram: the films showed no opacification of the gall-bladder at fourteen hours.

With the persistence of her pain, an ultra-sound scan of the abdomen on the 3rd of June subsequently showed a small thick-walled gall-bladder containing several medium-sized calculi. There was no associated dilation of the biliary tree. A formal diagnosis of

cholecystitis was made.

At this point, she was referred to the surgical unit. She still complained of a continuous, severe, gripping pain in the right upper quadrant and epigastrium which varied in intensity thoughout the day. It had no radiation but when at its worst, it made her feel nauseous and vomit. She also felt extremely nauseous after eating any sort of fried food.

In 1982, Mrs. S.G. developed hypertension. Her blood pressure had been raised during both her pregnancies. In February of 1983, she presented to the renal physicians in severe chronic renal failure - urea of 40 mmol/l and creatinine of 1.25 mmol/l. There was little in the way of reversible factors and, after being found to have small shrunken kidneys, she was started on maintenance haemodialysis.

On the 23rd of April, 1984, she received a successful cadaveric renal transplant, complicated by only one acute rejection episode six days post-operatively. This was successfully treated with haemodialysis and anti-rejection drugs.

At the time of referral to the surgical unit for cholecystectomy, her renal function was well maintained - urea of 7.0 mmol/l and creatinine of 0.10 mmol/l.

Mrs. S.G.'s medication at this time consisted of: Prednisolone, 10mg daily; Axathioprine, 150 mg daily; Hydrallazine, 25 mg twice daily; Moduretic, one tablet

daily; and Sando K, one tablet twice daily.

On the 26th of August, cholecystectomy was performed. Through a transverse sub-costal incision a small, shrunken gall-bladder was identified. The cystic duct was dissected out and an operative cholangiogram showed a normal calibre common bile duct with no filling defects and free flow into the duodenum. The gall-bladder was subsequently dissected free from the liver bed, the cystic duct tied and divided, and the operation completed with the wound being closed in layers.

Mrs. S.G. was well post-operatively and made a good recovery from the procedure. However, on the evening of 30th of August, she complained of the sudden onset of a sharp pain in her epigastrium and lower thorax. It was not pleuritic in nature and had no radiation but was associated with the vomiting of large amounts of bile-stained fluid. The pain was partially relieved by leaning forward.

Examination of Mrs. S.G.'s abdomen revealed only a tenderness to deep palpation in her epigastrium and around her cholecystectomy wound. She exhibited no guarding or rebound tenderness and was Murphy's sign negative. Bowel sounds were present and normal. The mass of her transplanted kidney could be felt in her left iliac fossa and was unchanged.

Neither of her calves were swollen or tender, and her ECG showed no acute changes.

The following morning, blood was sent to Clinical Chemistry:

		1.9.87	(Normal)
serum amylase	(u/l)	72	(50-300)
serum bilirubin	(μmol/l)	42	(3-14)
serum alkaline phosphatase	(u/l)	190	(30-140)
serum gamma-glutamyl transferase	(u/l)	155	(8-49)

It was concluded that she might have a stone in her common bile duct and on the 3rd of September, an ultra-sound scan of her upper abdomen was performed. It revealed her common bile duct to be slightly dilated at 9 millimetres and this was associated with some intra-hepatic dilation. Consequently, it was suspected that Mrs. S.G. had retained a stone in her common bile duct.

That evening, the resident house officer was called to see Mrs. S.G. She complained of very severe pain in her right and left upper quadrants similar in nature to before. She had vomited more bile-stained fluid.

On examination, she exhibited marked guarding and was tender even to light palpation of her epigastrium. Minimal bowel sounds were audible on auscultation.

It appeared likely that Mrs. S.G. had suffered a leakage of fluid from her common bile duct, producing a biliary peritonitis, and possibly associated with a

retained stone following her recent cholecystectomy. Her analgesia was increased and she was observed overnight.

Clinical Chemistry

		1.9.87	5.9.87
serum amylase		89	
serum bilirubin	(µmol/l)	63	73
serum alkaline phosphatase	(u/l)	203	321
serum gamma-glutamyl transferase	(u/l)	193	242

On the 7th of September, her abdominal pain started to radiate down into her right iliac fossa, giving the impression of fluid tracking inferiorly. On the 8th of September, she started to deteriorate and the medical registrar came to see her. Her abdominal pain was becoming worse and she was vomiting large amounts. Intra-venous fluids were commenced and and E.R.C.P. organised for the following day.

At E.R.P.C. (see Figure 10), a cavity was seen into which contrast initially leaked, leading to a second cavity into which it tracked more slowly. The anatomy of the biliary tree was poorly seen: only the left hepatic duct was filled by contrast.

From these findings it was concluded that either

(1) The stump of the cystic duct had opened up and that the right hepatic duct was not seen for

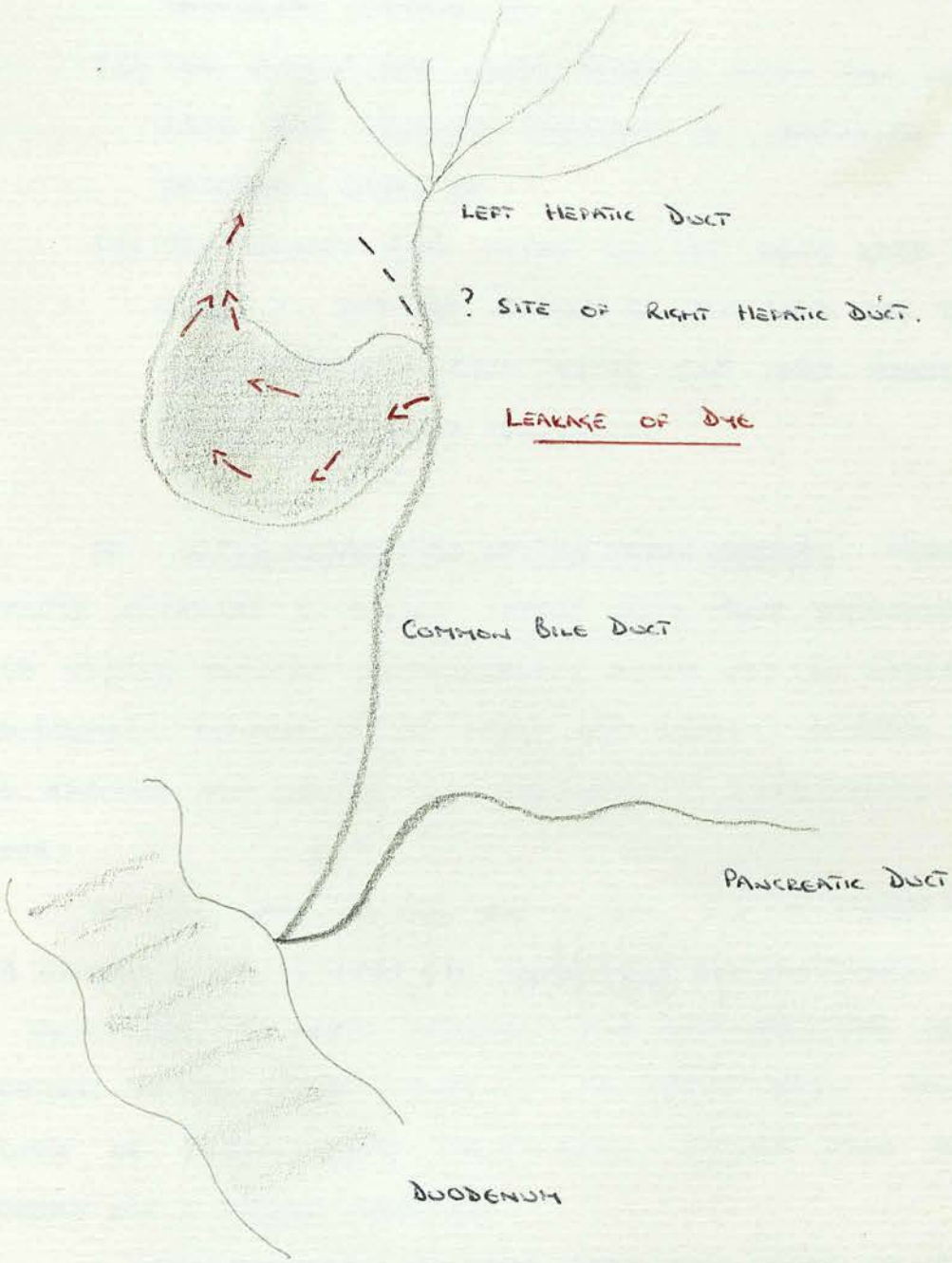

Figure 10: Diagram of ERCP showing leak of bile

technical reasons, or

(2) the cystic and right hepatic ducts had been tied and ligated together at operation to produce a leak, or

(3) the cystic duct stump had not been seen at E.R.C.P. and the source of the leak was the right hepatic duct which had been damaged during the recent operation.

An ultra-sound scan of the upper abdomen subsequently revealed a normal common bile duct associated with mildly dilated intra-hepatic ducts but no obvious sub-hepatic collection of fluid was seen. Ascites of the abdomen and pelvis was apparent, in addition to an ileus.

On the 10th of September, Mrs. S.G.'s condition had become quite serious and laparotomy was performed. At operation, a small oblique tear was detected just proximal to the intact stump of the cystic duct. Three litres of bile-stained fluid were drained from her abdomen and a T-tube inserted.

A cholangiogram did not show any stone in the common bile duct and the anatomy of the biliary tree was seen to be normal. In fact, the common bile duct appeared to be formed from three branches: the left hepatic duct and the medio-superior and latero-inferior branches of the right hepatic duct. This was assumed to be the reason for poor visualisation at E.R.C.P.

Mrs. S.G.'s condition improved markedly post-operatively. A cholangiogram performed after a week was normal, the T-tube was removed after ten days and she was subsequently discharged home.

Discussion

Biliary Peritonitis[1]: leakage from the biliary system into the peritoneal cavity most commonly follows disruption of its continuity: bile rarely leaks spontaneously.

Although bile irritates the peritoneum, and tends to become infected with organisms from the intestine, it may cause remarkably few symptoms even following a major leak. Usually there is poorly localised abdominal pain and shoulder pain on one or both sides. Often there are signs of peritoneal and diaphragmatic irritation, with muscle guarding. Low fever and tachycarida may present, with leucocytosis, electrolyte deficiency, dehydration and oliguria developing later. Occasionally, peritoneal irritation is such that large quantities of fluid collect, so called bilious ascites, or a sympathetic pleural effusion may develop with bile staining the fluid.

Billiary Injuries[2]: The commonest cause of injury to the biliary tract is mishaps during surgery. However, patients may occasionally present with injuries to the gall-bladder or bile duct resulting from blunt or penetrating trauma.

Trauma to the gall-bladder may be treated by repair or removal. Incomplete transection of the bile

duct may be repaired with T-tube drainage. However, complete division requires repair by anastomosis.

Accidental Lesions of the Common Bile Duct at Cholecystectomy:

Cholecystectomy is one of the most frequent operations performed in general sugery. Accidental lesions to the common bile duct are a complication of low frequency but they are always very serious, being associated with considerable morbidity and some mortality.[11] It has been described as "the most catastrophic complication of a straight-forward cholecystectomy".[3]

Andren-Sandberg et al studied all sixty-five reports of accidental lesions of the common bile duct reported in Sweden over a six year period from 1975-82,[4] to try to categorise avoidable factors and situations where the surgeon should pay extra attention.

Due to the relative infrequency of this complication of colecystectomy, studies of common bile duct injuries tend to be collected over a long time period, often up to thirty years;[5,6] these studies also tend to involve small numbers, and often include other types of injury, such as trauma by blunt or penetrating

injury, in addition to iatrogenic injuries.[7,8] An advantage of the study by Andren-Sandberg et al is the relatively short time period of seven years and the fact that all the cases from one country were included during that period. They found that:

(1) The type of patient offered little explanation for the occurrance of iatrogenic common duct lesions. They tended to be relatively young women (mean age 46 years compared with 54 years in the control group; 72 per cent of cases female), who were not overweight compared to their height, and wihout significant concomitant gastrointestinal or cardiopulmonary disease. None had undergone any previous abdominal surgery. The prerequisites for surgery were therefore good.

(2) The type of hospital did not affect the incidence of accidental lesions of the common bile duct, being as common in large university hospitals as small district hospitals.

(3) The experience of the surgeon was very important, with most injuries occurring at the hands of a surgeon who had previously performed between 25 and 100 cholecystectomies [see Table 3]. The relative lack of senior surgeons, resulted in 80 per cent of the cases which ended in bile duct injuries being performed by junior surgeons, who did not have much experience and who

Table 3 Earlier experience of cholecystectomies of the surgeon who made the lesion.[4]

No. of operations	No. of Surgeons
0 - 25	3
25 - 100	52
100 - 500	10
500	0

were often unsupervised. Andren-Sandberg et al suggested these surgeons had attained a routine to some degree but may have felt some over-confidence. They noted that the very inexperienced surgeon, who had performed less than twenty-five cholecystectomies, was normally supervised by a more experienced surgeon and so was less likely to damage the common bile duct.

These findings are in agreement with those of Lord Smith of Marlow,[5] who found that 85 per cent of iatrogenic common bile duct injuries occurred when a junior surgeon had been operating on his own [see Table 4].

Table 4: The Surgeon responsible for post-cholecystectomy bile duct stricture.[5] (Analysis of 400 patients from the United Kingdom selected at random)

Operation performed by	No. of Cases
: consultant	58
: registrar assisted by consultant	52
: registrar with consultant not present	338

(4) The urgency of operation: it was found that only two of the injuries (4 per cent) were done during acute operation. This is in contrast to a study by Castrini and Pappalardo[9] which observed a dominance of iatrogenic strictures following acute operations: 61 per cent compared with 42 per cent following elective operations.

(5) Detection: Andren-Sandberg et al described a perioperative detection rate of 85 per cent for iatrogenic lesions of the common bile duct. This is a marked improvement on 14 per cent in the previous study by Castrini and Pappalardo,[9] or 18 per cent in that by Hillis et al.[10] Results appear to be considerably better when the injury is detected and repaired immediately.[4]

Andren-Sandberg et al[11] found that 55 of the sixty-five lesions were detected and repaired at the cholecystectomy; the remaining 10 were detected and repaired during the first ten days after operation.

In 38 of the cases detected perioperatively, an end-to-end choledochostomy was performed. 22 per cent had a good result which did not require further surgical intervention.

The other 17 cases detected perioperatively were treated with choledocho - or hepatico enterostomy, and good results were achieved without surgical intervention

in 54 per cent.

Of the ten patients whose lesions were detected post-operatively, three received an end-to-end choledochostomy: all patients had their lesions repaired by choledocho- or hepatico enterostomy: three did not require further surgical intervention but four had to be reoperated on.

These results regarding end-to-end anastomosis compared to bilidigestive anastomosis must be considered with caution since the surgeon did not choose an operative approach at random. This Swedish report is less favourable than others reported in the literature:[6,7,8,9] this may be explained partly by the fact that the data is collected from an insurance syndicate, to which not all the favourable cases may have been reported.

It appears that end-to-end choledochostomy is not a good alternative, especially in delayed cases or cases where a portion of the common bile duct is lost, when Roux-en-Y hepaticojejunostomy might be a better alternative. Bismuth et al[12] found early complications in 12 of 101. However, this study was only of patients in whom the anastomosis was done in apparently healthy tissue.

Also, it appears that an early repair has certain advantages. In particular, there will always be an inflammatory process secondary to the bile accumulation

from a bile fistula, or obstruction of the duct, if a lesion is left for several days.[3]

Summary and Conclusions

Mrs. S.G. has suffered a relatively uncommon complication of cholecystectomy, which has resulted from damage to her common bile duct. Biliary peritonitis need not produce a particularly florid set of symptoms and signs, but in the case of Mrs. S.G. these may have been suppressed by the immunosuppressant treatment she was receiving. The long term outcome of her treatment remains to be seen.

Andren-Sandberg et al[4] recommend the better training of surgeons and better supervision of surgeons under training, as well as emphasis on good exposure and atraumatic surgery. Perioperative cholangiography should be performed to check that no unknown anomaly is present, as well as to search for bile duct stones.

There is a need for long-term follow up to assess the best treatment of common bile duct injury. It appears that cholodochoenterostomy should be the procedure of choice if freedom from complications during the first years is given highest priority.

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PART III

"LUMPS AND BUMPS"

Case 4

Mrs. A.B. is an 89 year old widow who presented to the surgical unit with a twenty-four hour history of a painful swelling in her right groin associated with vomiting. Her general practitioner sent her to hospital with a suspected incarcerated inguinal hernia.

She first noticed the swelling in her right groin the day before, while dressing in front of the mirror. It had since become increasingly painful and was now tender to the touch. Over this period, she had twice vomited a small bowl-full of yellow vomitus which had no signs of frank blood, "coffee-grounds" or mucus present.

Mrs. A.B. received a total hip replacement on her left side after suffering a fractured neck of femur. She had suffered from generalised osteo-arthritis for a number of years which limited her mobility.

On admission, she was receiving Frusemide, 40 mg in the morning; Thioridazine, 25 mg at night; and Codyromol, one tablet every six hours, if required.

She had spent the last four years living in a Church of Scotland home and it was one of the nursing staff from there who had provided most of the history. She had no significant family history and systematic enquiry was unhelpful.

On examination, Mrs. A.B. was mildly distressed and disorientated as to where she was. She showed some signs of dehydration and mild pitting oedema of her ankles. However, her cardiovascular system was stable and her chest was clear.

Her abdomen was distended but it was soft with no tenderness, guarding or masses except in her right groin. There was no organomegaly.

Examination of her right groin revealed a swelling, approximately 5 centimetres by 3 centimetres. The skin overlying this swelling was normal looking. On palpation, this tender groin swelling was found to be soft, irreducible and mobile. On auscultation there were no bowel sounds audible over the mass but bowel sounds in the rest of the abdomen were increased in pitch.

With Mrs. A.B. lying down, the relationship of this swelling to the inguinal ligament and pubic tubercle was not entirely clear, but as soon as Mrs. A.B. was helped to her feet, it was clear that it lay below and lateral to the tubercle. No cough impulse could be felt over the inguinal ring.

Clinical chemistry confirmed that she was dehydrated when her blood urea was found to be 13.5 mmol/l with a normal creatinine level.

Abdominal X-ray showed moderately dilated loops of small bowel entirely consistent with a degree of mechanical obstruction, as suggested by clinical

examination [see Figure 11].

A diagnosis of incarerated femoral hernia was made and Mrs. A.B. was prepared for theatre. At operation, a McEvedy's approach was made to the femoral canal. The pre-peritoneal space was developed and the femoral sac reduced without difficulty. On opening the sac it was found to contain no small intestine but rather only omentum. The surrounding small bowel was seen to normal.

The neck of the sac was transfixed, the femoral canal slenderized and the wound closed.

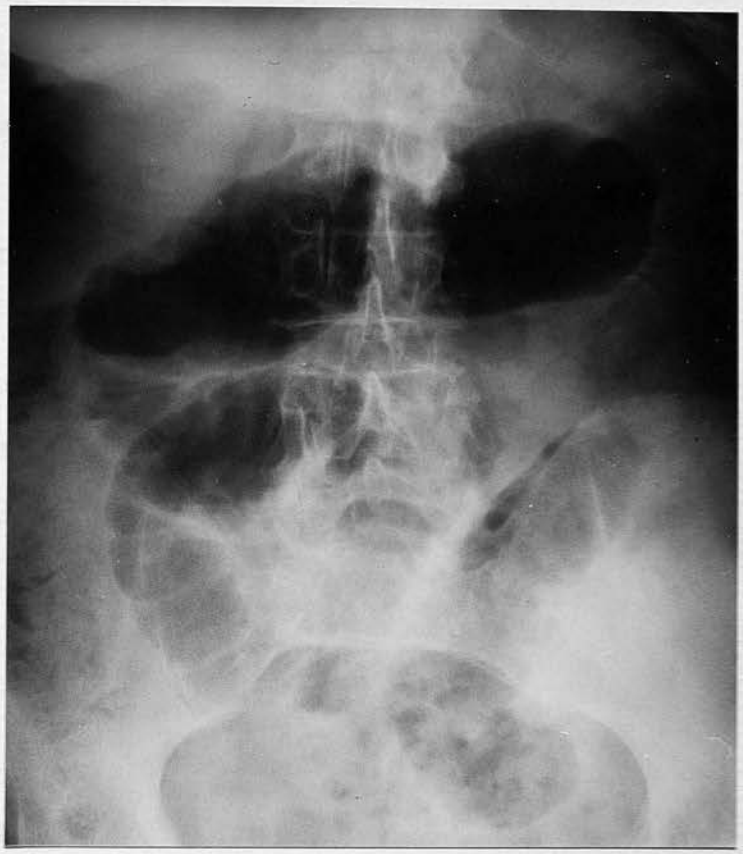

Figure 11: Plain abdominal X-ray showing moderately dilated loops of small bowel

Discussion

A hernia is a swelling caused by the protrusion of part of an organ, or other tissue, through an aperture in the walls of its retaining space.

A femoral hernia is the protrusion of a peritoneal sac through the femoral canal, medial to the femoral vessels as they pass under the inguinal ligament. It is often covered with extraperitoneal fat and usually contains omentum, small bowel or both.

Femoral hernia accounts for 20 per cent of hernias in women but only 5 per cent of hernias in males. This difference is due to the stretching of the pelvic ligaments and resulting widening of the femoral ring during pregnancy.

The femoral canal occupies the most medial compartment of the femoral sheath, and it extends from the femoral ring above to the saphenous opening below. It normally contains fat, lymphatic vessels and the lymph node of Cloquet.

The femoral ring is bounded anteriorly by the inguinal ligament, laterally by a thin septum separating it from the femoral vein and medially by the edge of the lacunar ligament. The pectineal ligment of Cooper, the pubic bone and the fascia over the pectineus muscle lie posteriorly.

A femoral hernia pushes the extraperitoneal fat and peritoneum through the femoral ring and down the femoral canal. On leaving the canal the hernia turns superficially to pass through the saphenous opening of the deep fascia of the thigh. Pushing the cribriform fascia in front of it, the hernia turns upwards to lie over the inguinal ligament.

Clinical Features: A femoral hernia typically presents as a bulge in the upper inner aspect of the thigh just beneath the inguinal ligament. On emerging from the saphenous opening it forms a well-defined, soft or firm swelling which is situated below and lateral to the public tubercle but which may extend upwards over the inguinal ligament. At this stage it may be difficult to distinguish from an inguinal hernia but the correct diagnosis is suggested by the absence of a cough impulse over the inguinal ring and the fact that a femoral hernia is difficult to reduce due to its tortuous course.

However, relationship to the pubic tubercle is the key to diagnosis. A femoral hernia emerges below and lateral to the pubic tubercle while an inguinal hernia emerges above and medial to it.

Femoral hernia is very rare before the age of fifteen but the prevalence rises after twenty years of age and continues to rise into old age. The right side is affected twice as often as the left, and it is

bilateral in 20 per cent of cases. The symptoms of a femoral hernia are not pronounced and it may go unnoticed for some time.

Differntial Diagnosis: A femoral hernia has to be distinguished from:

(1) an inguinal hernia,

(2) a saphenous varix,

(3) enlarged lymph nodes,

(4) a lipoma.

Also, a femoral hernia may be confused with a femoral aneurysm, a psoas abscess, a distended psoas bursa or a rupture of the adductor longus with haematoma formation.

Treatment: The overriding importance of femoral hernia lies in the fact that it cannot be controlled by a truss, and that of all hernias, it is most likely to become strangulated.

The principles of the operation are complete excision of the sac and repair of the defect (the femoral canal is "slenderised"). There is no operation for femoral hernia which is ideal or uniformly applicable. If strangulation has occurred, access is required to the main abdominal cavity. Three approaches to femoral herniorrhaphy have been described:

(1) the crural or "low" operation of Lockwood (1893);

(2) the inguinal or "high" operation of Annandale (1875) and Lotheissen (1898);

(3) the abdominal, suprapubic or extraperitoneal operaton: this was developed by Henry (1945) with a midline incision and later by McEvedy (1950) using a pararectal incision.

Following reduction and excision of the hernial sac, the inner surface of the conjoint tendon is sutured to the pectineal ligament of the pubis.

A McEvedy's approach is preferred if there is any hint of strangulation and also gives best access to the femoral ring. Lotheissen's operation has the drawbacks that it disrupts the inguinal canal mechanism and does not provide adequate access to a strangulated viscus. The "low" approach is bloodless and repair of the hernia easy. However, its access to a strangulated hernia is often very inadequate.

Summary and Conclusions

Mrs. A.B. complained of the swelling in her groin for twenty-four hours on presentation, but clearly it may have been there for some time. The mistaken diagnosis made by her general practitioner demonstrated the importance of proper examination of the groin and correct interpretation of the anatomical land-marks.

The clinical history of vomiting and the moderately dilated loops of the small bowel seen on plain abdominal X-ray suggested that a partial, if not complete, mechanical obstruction had recently occurred. However, the hernial sac at operation contained no bowel. It was assumed that under the influence of general anaesthetic, loosening of the surrounding tissues had allowed the piece of bowel caught in the sac to fall back into the peritoneal cavity. The small bowel was seen to be normal and no further action was required.

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Case 5

Mr. R.C., a 23 year old male who presented with a three month history of a painless lump in his right testicle which had been gradually increasing and had now reached the size of a golf ball.

He reported no abnormality in his genitourinary system; specifically, no dysuria, haematuria or increase in frequency of miturition and no difficulty with sexual intercourse. He had not had any operations or serious illnesses in the past, and had no history of trauma to his scrotum.

Mr. R.C. had been feeling well over the past three months, was not receiving any medication and did not know of anything to which he was allergic. He drank alcohol socially with his friends at the weekends but was a non-smoker. He was married, without any children but hoped to start a family in the future; he worked as a guard for British Rail.

Systemic enquiry was unrevealing.

On examination, Mr. R.C. was a healthy looking man. The only abnormality was in his scrotum. On inspection, a swelling could be seen on the right side of his scrotum but the skin overlying this swelling appeared normal with no sign of inflammation. A hard, non-tender, ovaloid mass could be felt attached to the antero-inferior surface of the right testis. Only a

small rim of normal testis could be felt superiorly. The mass measured approximately 5 x 3 x 3 centimetres and did not transilluminate.

The left testis was clinically normal and the inguinal nodes were not enlarged.

At out-patients, it was considered that this lump was a testicular tumour and it was arranged for Mr. R.C. to be admitted the following day for orchidectomy.

On the 13th of October, an ultra-sound scan of both testes and the para-aortic region was performed. This revealed a small rim of normal tissue at the upper pole of the right testis, with the remainder almost completely replaced by a large solid mass of mixed echoity which was thought to be a tumour, possibly a teratoma. The left testis appeared normal. The liver was also scanned and appeared normal, with no evidence of metastatic disease.

A chest X-ray taken the same day demonstrated clear lung fields. Blood taken for tumour markers was found to support the diagnosis of a testicular carcinoma.

		13.10.87	(Normal Value)
plasma alpha-fetoprotein	(u/ml)	163	(2-6)
plasma beta-human chorionic chorionic gonadotrophin	(mIU/ml)	17	(<10)

The other haematology and clinical chemistry tests were normal. A mid-stream specimen of urine produced no growth on culture.

On the 16th of October, Mr. R.C. was taken to theatre for a right orchidectomy with insertion of a testicular prostheis. Biopsy of the left testicle was also performed.

The right testis was approached through an inguinal incision and delivered into the wound. The testis was removed after clamping and ligation of the spermatic cord and sent fresh to the laboratory for histopathological analysis. A medium-sized sialastic testicular prostsis was then inserted and the wound closed in layers.

On the left side, a small incision was made over the testicle, through which it was inspected. Its surface appeared normal and a testicular biopsy was taken to search for carcinoma-in-situ.

In Pathology, the right testicular tumour was seen to be a multilobulated mass, measuring 5 centimetres in diameter and replacing most of the testicular parenchyma. Small cysts were present in parts of the tumour but no foci of haemorrhage or necrosis were identified.

Histology revealed a mixed testicular tumour

composed of both seminoma and teratoma but there was no invasion oof the rete testis or spermatic cord. The teratoma showed an admixture of well-differentiated organoid areas and embryonal areas resembling yolk sac elements. No foci of chorio-carcinoma were identified [see Figure 12].

The testicular tubules immediately adjacent to the tumour were compressed and hypoplastic but elsewhere showed normal spermatogenesis with no evidence of carcinoma-in-situ (intra-tubular germ cell neoplasia).

The biopsy of the left testis also showed no evidence of carcinoma-in-situ.

Immuno-peroxidase stains for tumour markers indicated that both alpha-fetoprotein and placental alkaline phosphatase were abundant within the tumour although chorionic gonadotrophins were not identified.

On discharge, Mr. R.C.'s testicular tumour had been successfully removed and he had no evidence of either carcinoma-in-situ in his left testis from biopsy, or of metastatic disease from an ultra-sound scan of his abdomen and a chest X-ray. In addition, his serum tumour markers were continuing to fall at a rate consistent with their half-life and so, at the Department of Radioation Oncology where he was sent for follow-up, he did not receive any immediate chemotherapy treatment.

On the 29th of October, CT scans of his thorax and abdomen showed no signs of metastatic disease and his

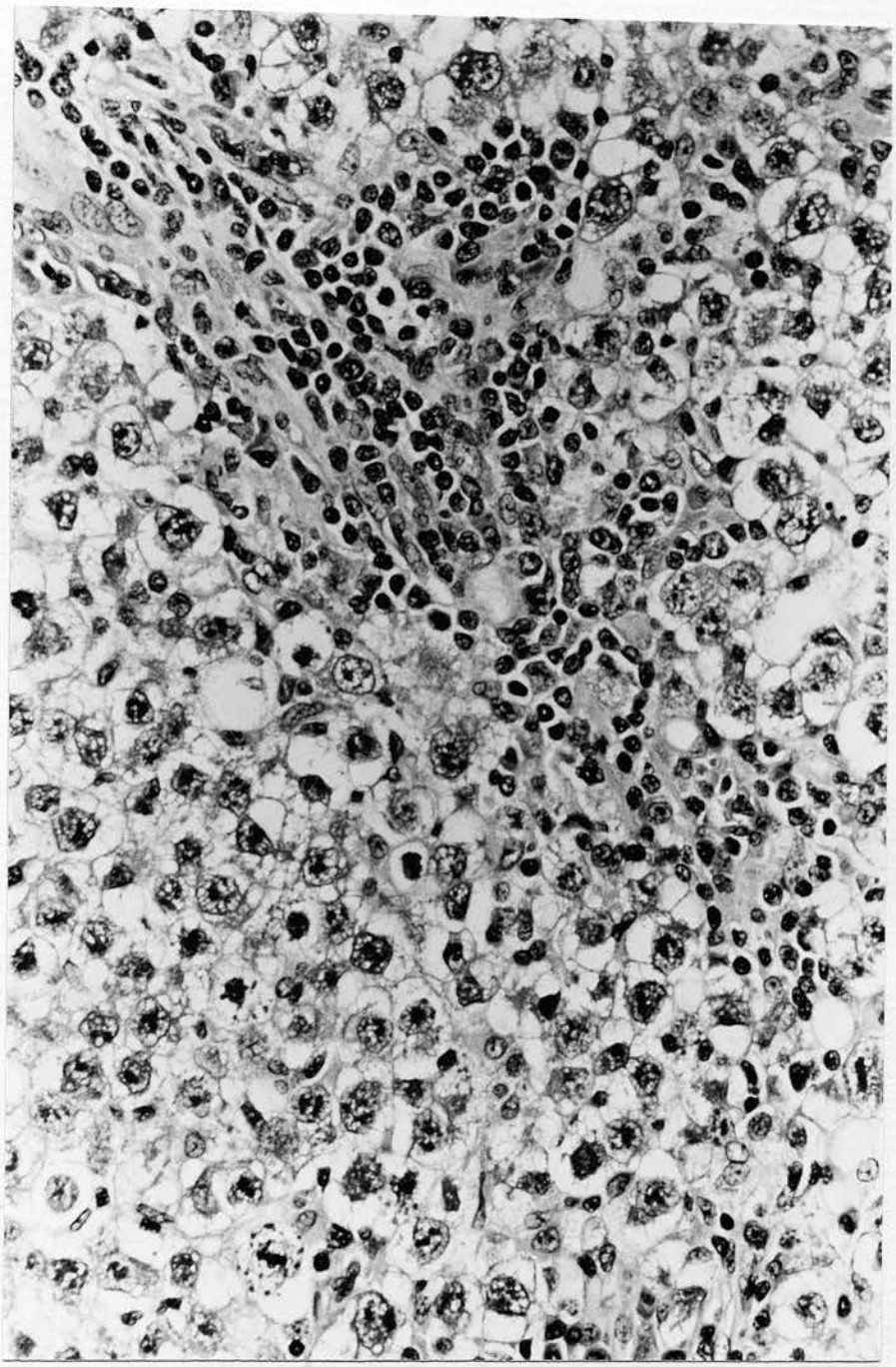

Figure 12i: High power picture showing seminomatous component of tumour

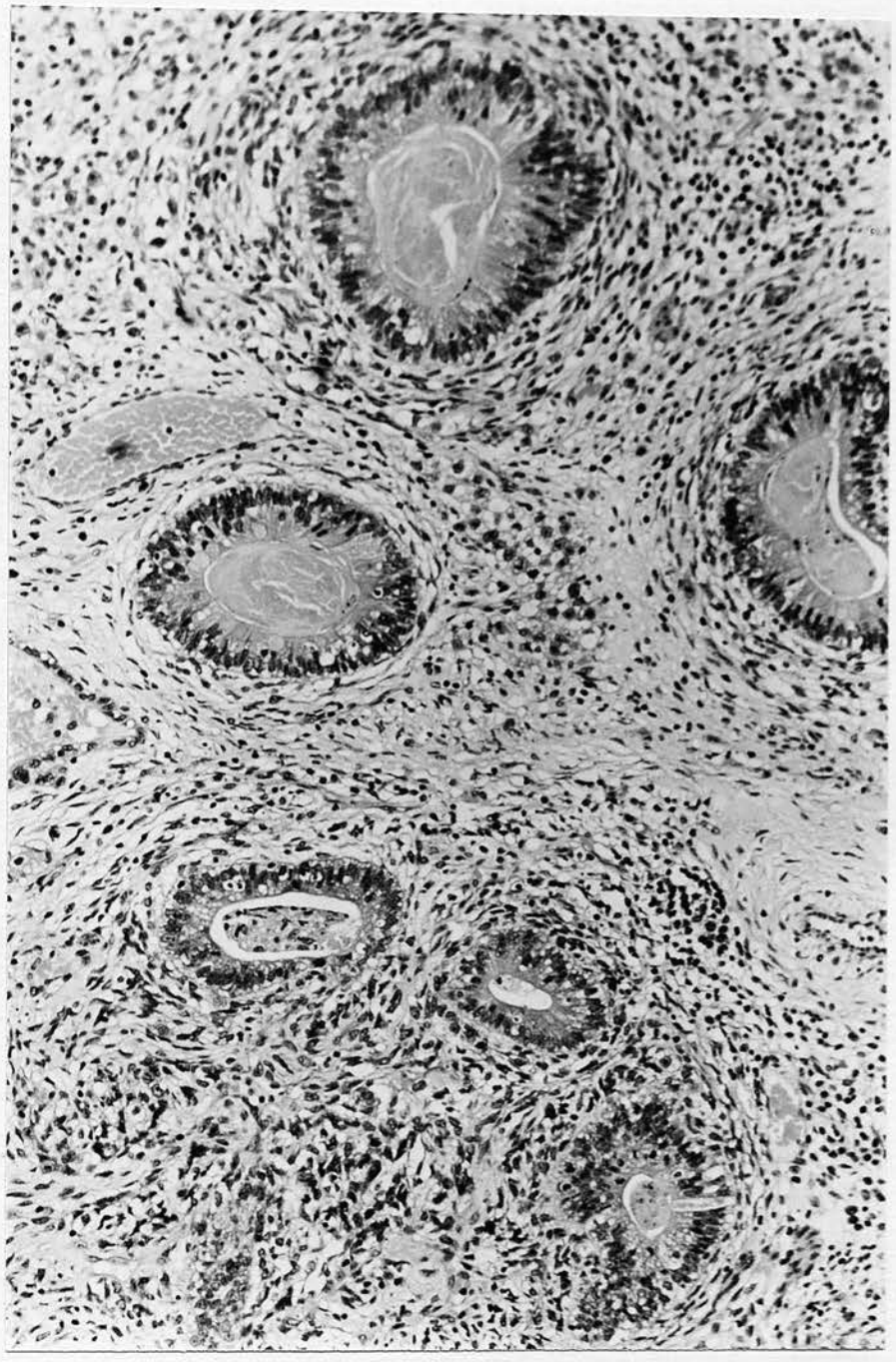

Figure 12ii: Teratoma showing glandular differentiation

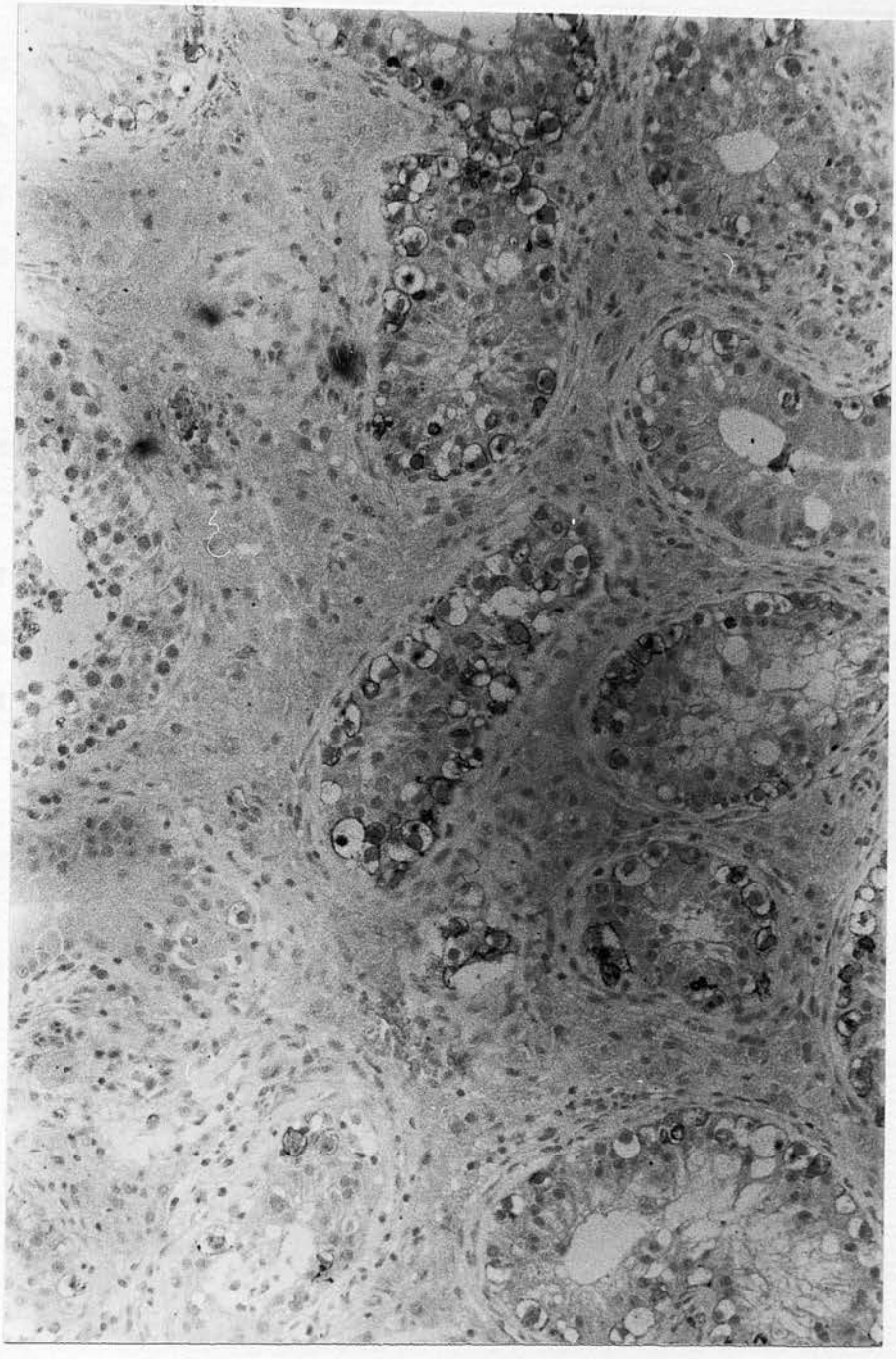

Figure 12iii: Cells stained for placental alkaline phosphatase

serum tumour markers were consistent with this [see Table 5].

Mr. R.C. was put on an observation programme of monthly follow-up appointments for one year. Every month he will have his serum tumour markers checked and every four months he is to have a CT scan, to search for signs of metastic disease.

It was explained to Mr. R.C. that there was a 20 per cent chance of recurrance and if this were to occur, that he would subsequently receive chemotherapy. However, on the 1st of February, CT scans showed no evidence of any tumour growth and his condition was encouraging.

Table 5

		13.10.87	19.10.87	9.11.87	7.12.87	11.1.88	8.2.88	7.3.88
alpha-fetoprotein	u/ml (2-6)	163	93	7	2	2	2	1
beta-human chorionic gonadotrophin	(less than 5)	17	3	3	Less than 3	Less than 3	Less than 3	Less than 3
lactate dehydrogenase	u/1 (72-395)			500	420	505	363	495
aspartate amino transferase	u/1 (9-52)				33	59	37	38

half-life of alpha-fetoprotein : 5 to 7 days
half-life of beta-human chorionic gonadotrophin : 1 to 2 days

Discussion

Germ cell tumours of the testis are unusual in the field of oncology for a number of reasons. Firstly, their incidence has been rising so that, in developed coutries, they are now the most common tumours in men between the ages of 25 and 34 years. However, in parallel to this, prognosis has dramatically improved over the last 15 years so that 90 to 95 per cent of patients with testicular tumous are now cured.[1]

The aetiology of testicular tumours is unknown but they are more common in men with testicular maldescent. This risk relates to the degree of maldescent and so is much higher in an abdominal testis. The normal descended contra-lateral testis is also at increased risk of tumour, which suggests a background abnormality associated with maldescent rather than the abnormal location of the testis causing the increassed risk.[1]

The most common sign of a testicular tumour is a hard, enlarged testis. Other conditions which should be considered as differential diagnosis include epididymitis, torsion of the testis, tuberculosis and graulomatous orchitis. Diagnosis is confirmed at inguinal orchidectomy. As breach of the tunica albuginea by the tumour may predispose to local recurrance, scrotal incisions are usually avoided at

operation.

(1) Seminomas are described as classical or anaplastic, although both have a similar natural history. They arise from the seminiferous tubules and are of relatively low grade malignancy. Seminomas are very sensitive to radiotherapy.

(2) Teratomas (non-seminomatous tumours) arise from primitive germinal cells and may contain cartilage, bone, muscle, fat and a variety of other tissues. They are classified according to the degree of differentiation.
   (a) teratoma differntiated (teratoma)
   (b) malignant teratoma intermediate (teratocarcinoma)
   (c) malignant teratoma undifferntiated (embryonal carcinoma)
   (d) malignant teratoma trophoblastic (choriocarcinoma)

(3) Combined tumour contains both seminoma and teratoma.

After diagnosis of a testicular tumour by orchidectomy, assessment should provide information on the extent and bulk of metastatic disease, the presence

and levels of serum tumour markers and the general health of the patient.

Royal Marsen Hospital staging of testicular tumours

Stage I	No evidence of metastases
I+	Rising serum markers with no other evidence of metastesis
II	Abdominal node involvement
III	Supradiaphragmatic node involvement
IV	Extra-lymphatic metastases

Tumour markers are helpful in the management of patients with germ cell tumours. Following orchidectomy, they can provide valuable information on the presence of metastases. In stage I disease, following orchidectomy, serum concentrations of the tumour markers should fall at a rate consistent with their physiological half-life: the half-life of alpha-fetoprotein is 5 to 7 days while that of beta-human chorionic gonadotrophin is 1 to 2 days. A slower rate of fall, level concentrations, or a rise in markers is likely to be due to marker production by metatstic disease.

Carcinoma-in-situ, or premalignant cells, may be present in the contralateral testis of men with testicular tumours.[2] The detection of these cells would probably allow an early cancer to be cured.[3]

Testicular biopsy is the only reliable way to detect carcinoma-in-situ, although the abnormal cells may be missed. However, there seems to be a spread of premalignant cells throughout the testis so that they are likely to be detected by random biopsy.[4] Diagnosis also depends on good specimen preparation, and a new development is using immunoperoxidase and antibodies against placental alkaline phosphatase which appears to stain carcinoma in situ cells as well as seminoma cells.[3]

Carcinoma-in-situ is most common in the seminiferous tubules adjacent to a germ cell tumour but may be present in approximately 5 per cent of contralateral testes.[5] Adult men with a history of madescent of the testis,[6] infertile men[7] and men with gonadal dysgenesis are also at risk.[8]

The risk of carcinoma-in-situ progressing to a germ cell tumour is high: in a London series of infertile men, eight were found to have carcinoma-in-situ on biopsy and six subsequently developed tumours.[9]

Von der Masse et al[5] recommended that surgeons should take a biopsy specimen from the contra-lateral testes of all men undergoing orchidectomy for testicular tumour. Men with metastases who will receive chemotherapy anyway, probably do not need biopsy, but when orchidectomy is to be followed only by surveillance in men with early stage germ cell tumours, the surgeon should consider biopsy of the contralateral testes.

Conclusions

In the case of Mr. R.C., his tumour was judged to be at an early stage from clinical examination, chest radiography and abdominal ultrasonography. It was thought to be localised at orchidectomy so, in line with the policy of the hospital,[10] a biopsy was taken from the contralateral testis and Mr. R.C. was put on surveillance.

A reluctance to search for carcinoma-in-situ may be due, in part, to a lack of awareness of the condition and partly because the true risk of progression is only now becoming apparent.[10]

Standard treatment of carcinoma-in-situ remains orchidectomy, but low dose radiotherapy may be an option for men with bilateral carcinoma-in-situ or carcinoma-in-situ in the single remaining testis.[11]

As a result of carcinoma-in-situ of the testis being clearly described and the men at risk being described, it should be possible to prevent a large proportion of the resultant tumours and to reduce the number of men with metastatic disease.

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